Environmental Exposure to Benzene: An Update

Lance Wallace

U.S. Environmental Protection Agency, Reston, Virginia

During the 1990s, several large-scale studies of benzene concentrations in air, food, and blood have added to our knowledge of its environmental occurrence. In general, the new studies have confirmed the earlier findings of the U.S. Environmental Protection Agency Total Exposure Assessment Methodology (TEAM) studies and other large-scale studies in Germany and the Netherlands concerning the levels of exposure and major sources. For example, the new studies found that personal exposures exceeded indoor concentrations of benzene, which in turn exceeded outdoor concentrations. The new studies of food concentrations have confirmed earlier indications that food is not an important pathway for benzene exposure. The results of the National Health and Nutrition Examination Survey on blood levels in a nationwide sample of 883 persons are in good agreement with the concentrations in exhaled breath measured in about 800 persons a decade earlier in the TEAM studies. Major sources of exposure continue to be active and passive smoking, auto exhaust, and driving or riding in automobiles. New methods in breath and blood sampling and analysis offer opportunities to investigate short-term peak exposures and resulting body burden under almost any conceivable field conditions. — Environ Health Perspect 104(Suppl 6):1129–1136 (1996)

Key words: benzene, exposure, indoor air, outdoor air, personal monitors, TEAM study, body burden, breath, blood

Introduction

Much of our knowledge of nonoccupational exposure to benzene was supplied throughout the 1980s by the U.S. Environmental Protection Agency (U.S. EPA) Total Exposure Assessment Methodology (TEAM) studies of volatile organic compounds (VOCs) (*1*). These studies employed personal air quality monitors to measure direct personal exposures of approximately 800 persons in about eight areas in the United States between 1980 and 1987 (*2–13*). The participants were selected on a strict probability sampling basis to represent about 800,000 persons in these areas. Measurements of indoor and outdoor air, drinking water, and exhaled breath were made to supplement the personal air measurements. In a pilot study (*2,14*), measurements were also made in food and beverages; since few VOCs and no benzene was detected, those measurements were not repeated in the main study.

The basic results of the TEAM study as they apply to benzene may be summarized as follows (*15–20*):

- Benzene was not found, or was found in insignificant amounts, in water, food, and beverages. More than 99% of the total personal exposure was through air.
- Mean personal air exposures exceeded indoor air concentrations, which in turn exceeded outdoor air concentrations. A global average personal exposure was about 15 $\mu g/m^3$ (range 7–29 $\mu g/m^3$). Indoor concentrations were measured only in the 1987 TEAM studies in Los Angeles, CA, Baltimore, MD, and Bayonne, NJ, and appeared to be on the order of 10 $\mu g/m^3$. Outdoor concentrations had a global average of 6 $\mu g/m^3$ (range 2–19 $\mu g/m^3$).
- No effect on personal exposure of living close to major fixed sources of benzene (oil refineries, storage tanks, chemical plants) could be detected in Beaumont, TX (*2,3*); Bayonne and Elizabeth, NJ (*5–8*); or Los Angeles, Antioch, and Pittsburg, CA (*9–11*).
- The overwhelming source of benzene exposure for smokers was mainstream cigarette smoke (*15*). Smokers had an average benzene body burden about 6 to 10 times that of nonsmokers, and received about 90% of their benzene exposure from smoking (Figure 1). Roughly half the total benzene exposure in the United States was borne by smokers.
- For nonsmokers, most benzene exposure ultimately is derived from auto exhaust or gasoline vapor emissions. This includes most of the benzene exposure due to outdoor air, indoor exposures due to intrusion of evaporative gasoline fumes from attached garages (*21*), and personal activities such as driving (Figure 2). A portion of the exposure is due to environmental tobacco smoke (*15*). A small portion (about 6%) of the exposure is due to major point sources of benzene, such as petrochemical plants or refineries.

Two large-scale European studies (*22,23*) confirmed the TEAM study results for indoor and outdoor benzene concentrations. The study in Germany (*23*) also confirmed the effect of environmental tobacco smoke, finding an increase of 4.5 $\mu g/m^3$ in homes with smokers compared to the TEAM study finding of an increase of 3.5 $\mu g/m^3$. Both results were based on about 200 homes with smokers and 300 homes without smokers.

Recent Studies

About half-a-dozen large-scale studies of personal or indoor air levels of benzene

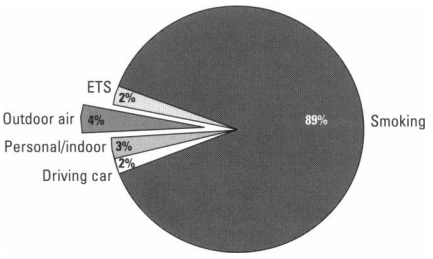

Figure 1. Sources of benzene exposure: smokers. A typical smoker takes in roughly 2 mg benzene/day; about 1.8 mg is delivered by mainstream smoke (55 μg/cigarette × 32 cigarettes per day). Source: U.S. EPA TEAM studies.

This paper was presented at Benzene '95: An International Conference on the Toxicity, Carcinogenesis, and Epidemiology of Benzene held 17–20 June 1995 in Piscataway, New Jersey. Manuscript received 16 January 1996; manuscript accepted 14 June 1996.

The research described in this article has been funded in part by the U.S. Environmental Protection Agency under contract 68-02-4544 to the Research Triangle Institute. It has been subjected to U.S. EPA review and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

Address correspondence to L. Wallace, U.S. EPA, 11568 Woodhollow Court, Reston, VA 20191. Telephone: (703) 341-7509. Fax: (703) 341-7575. E-mail: wallace.lance@epamail.epa.gov

Abbreviations used: NHANES, National Health and Nutrition Examination Survey; TEAM, Total Exposure Assessment Methodology.

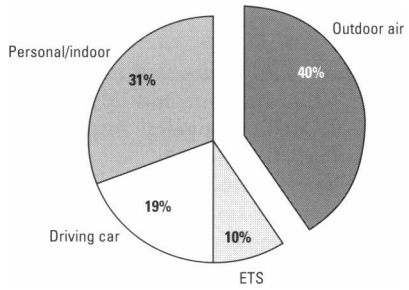

Figure 2. Sources of benzene exposure: nonsmokers. A typical nonsmoker inhales about 0.2 mg benzene/day, assuming an average exposure of 15 μg/m^3 and an alveolar respiration rate of 14 m^3/day. Outdoor air contributes about 40% of that amount, assuming an average outdoor level of 6 μg/m^3. The remaining 9 μg/m^3 are split between driving (100 min at 30–40 μg/m^3), indoor sources such as automobile vapor emissions in attached garages or storage of gasoline or kerosene in the garage or the basement, and environmental tobacco-smoke exposures at home or at work. Source: U.S. EPA TEAM studies.

have been conducted since 1990. They are briefly described below.

Personal Exposure Studies

A 1991 study (*24*) took place in 128 homes in Woodland, California, a community in a largely agricultural region. Personal, indoor, and outdoor benzene concentrations were measured using both Tenax (Enka Research Institute, Arnhem, the Netherlands) and evacuated canister samplers. Good agreement was noted between the side-by-side Tenax and canisters. Mean concentrations were 5.0, 4.0, and 1.2 μg/m^3 for the personal, indoor, and outdoor samples.

Day and night 12-hr average concentrations of benzene were measured for 58 residents of Valdez, Alaska (*25*). The mean benzene concentrations in the personal, indoor, and outdoor samples were 20, 16, and 5 μg/m^3 during the summer, and 28, 25, and 11 μg/m^3 during the winter, respectively.

Personal exposures to benzene were measured over a 3-hr period in the evening for 49 nonsmoking females in Columbus, Ohio (*26*). The median value in 25 homes with a smoker was 4.0 μg/m^3 compared to 2.4 μg/m^3 in 24 homes without smokers. The difference was statistically significant.

Personal exposures to benzene as measured in the TEAM studies and in the Valdez and Woodland studies are summarized in Table 1. Outdoor and indoor benzene values in the TEAM, Valdez, and Woodland studies are summarized in Tables 2 and 3.

Indoor Air Studies

A nationwide Canadian study (*27*) measured 24-hr indoor air concentrations of benzene in 754 randomly selected homes. Benzene mean indoor concentrations were 6.39, 5.60, 2.72, and 6.98 μg/m^3 in the winter, spring, summer, and fall seasons, respectively.

Indoor and outdoor 48-hr average concentrations of benzene were measured at 161 homes throughout much of California (*28*). The Pro-Tek charcoal badges formerly manufactured by E.I. duPont (Newark, DE) were used. Indoor mean concentrations were 8.3 μg/m^3 compared to 6.1 μg/m^3 outdoors.

Seventeen volunteers in Windsor, Canada, wore 3-stage adsorbent tubes with pumps in three microenvironments: at home, at work, and during commuting (*29*). Benzene concentrations were 3.5, 4.7, and 15.7 μg/m^3 in these three locations during summer 1991 and 2.7, 2.7, and 15.1 μg/m^3 during winter 1992. Outdoor levels near homes were 3.8 and 2.0 μg/m^3 during summer and winter, respectively. A later study (summer 1992) considered various microenvironments. Benzene levels averaged 2.2 μg/m^3 in homes of 26 asthmatics, 4.6 μg/m^3 in 13 samples from hotel rooms, 6.0 μg/m^3 in 17 samples collected during commuting, 20.8 μg/m^3 in 39 samples from four bingo halls, and 34.5 μg/m^3 in two taverns.

Brown and Crump (*30*) reported on a study of 173 homes in Avon, England. Passive Tenax tubes (Perkin-Elmer) collected

Table 1. Personal air concentrations (μg/m^3) of benzene measured in the TEAM, Valdez, and Woodland studies.

Site	Household, estimated no.	Year, season	Time	*n*	Mean	SE	Geom mean	Percentile 25	50	75	90	95	Max
NJ1	130,000	1981, fall	Day	340	26.2	2	11	7	17	32	65	81	270
			Night	347	29.7	5	13	7	15	32	54	73	510
NJ3	Unweighted data	1983, winter	Day	47	21.0	2	16	9	16	26	46	62	64
			Night	49	16.6	1	13	9	14	24	29	32	47
GNC	130,000	1982, spring	Day	24	7.9	2	8	–	8	13	–	–	36
			Night	24	10.2	2	12	–	12	16	–	–	43
A-P	91,000	1984, spring	Day	67	8.5	1	7	5	6	11	17	21	25
			Night	69	6.5	1	5	2	4	8	16	18	32
LA1	360,000	1984, winter	Day	112	19.1	2	15	10	15	23	35	51	86
			Night	112	16.5	1	14	11	15	21	30	34	43
LA2	330,000	1984, summer	Day	50	10.5	2	7	3	7	12	25	34	54
			Night	50	7.8	1	5	2	4	9	25	29	35
LA3	Unweighted data	1987, winter	Day	33	21.6	6	13	7	13	221	40	139	163
			Night	32	13.6	2	10	6	12	19	22	32	42
LA4	Unweighted data	1987, summer	Day	40	13.7	3	9	5	7	13	26	84	98
			Night	40	7.1	1	5	4	5	8	16	22	26
BAL	70,000	1987, spring	Day	70	16.4	2	9	–	11	22	32	45	129
			Night	70	20.0	3	12	–	14	24	42	62	104
VAL	Unweighted data	1990, summer	Day	55	25.4	5	14	7	13	23	70	130	210
			Night	58	15.5	3	9	5	9	20	30	70	130
VAL	Unweighted data	1991, winter	Day	56	34.4	6	21	13	20	37	90	110	230
			Night	58	23.6	4	13	7	12	26	65	100	170
WDL	30,000	1990, spring	24 hr	93	5.0	1	3	2	3	5	9	–	46

Abbreviations: *n*, number; SE, standard error; geom mean, geometric mean. NJ1, Bayonne–Elizabeth, NJ; NJ3, Bayonne–Elizabeth, NJ; GNC, Greensboro, NC; A-P, Antioch–Pittsburg, CA; LA1, Los Angeles, CA; LA2, Los Angeles, CA; LA3, Los Angeles, CA; LA4, Los Angeles, CA; BAL, Baltimore, MD (Dundalk); VAL, Valdez, AK; WDL, Woodland, CA.

Table 2. Household-weighted outdoor air concentrations (μg/m³).

Site	Households, estimated no.	Year, season	Time	*n*	Mean	SE	Geom mean	Percentile 25	50	75	90	95	Max
NJ1	40,000	1981, fall	Day	88	9.5	0.9	3.8	1.2	7.8	16	20	27	44
			Night	84	8.6	1	4.1	2.2	6.7	11	15	24	91
NJ3	Unweighted data	1983, winter	Day	8	3.9	0.8	–	2	4	5.4	7.3	–	7.3
			Night	9	4.3	0.7	–	2.6	4.5	5.5	7.9	–	7.9
A-P	25,000	1984, spring	Day	10	2	0.6	1.5	0.9	13	1.6	6.3	–	6.3
			Night	10	1.8	0.3	1.6	1.4	1.7	1.9	3.2	–	3.6
LA1	120,000	1984, winter	Day	24	13	1.3	11	8.1	14	18	21	22	35
			Night	24	19	1.9	16	11	19	25	32	33	33
LA2	110,000	1984, summer	Day	24	4.2	0.8	3.2	2	3.1	4.8	8.7	12	15
			Night	23	3.1	0.4	2.6	1.7	2.5	4.4	5.8	6.7	8.5
LA3	Unweighted data	1987, winter	Day	41	4.7	0.5	3.8	2.6	3.8	6.2	8.7	12	14
			Night	46	9.6	1	6.7	3.6	7.9	15	19	24	25
LA4	Unweighted data	1987, summer	Day	38	3.4	0.4	2.8	1.9	2.6	4.8	6.6	8.7	12
			Night	40	4	0.5	3.2	2	3.3	4.5	9	11	15
VAL	Unweighted data	1990, summer	Day	30	4.8	0.4	4.1	3	5	7	8	9	10
			Night	28	5	1	3.6	2	5	7	10	11	14
VAL	Unweighted data	1991, winter	Day	29	15	3.4	10	7	11	15	27	29	100
			Night	28	8.4	0.9	7	5	8	12	15	16	18
WDL	10,000	1990, spring	24 hr	48	1.2	0.9	1.1	0.8	1.1	1.4	1.9	–	3

Abbreviations: *n*, number; SE, standard error; geom mean, geometric mean. NJ1, Bayonne–Elizabeth, NJ; NJ3, Bayonne–Elizabeth, NJ; A-P, Antioch–Pittsburg, CA; LA1, Los Angeles, CA; LA2, Los Angeles, CA; LA3, Los Angeles, CA; LA4, Los Angeles, CA; VAL, Valdez, AK; WDL, Woodland, CA.

Table 3. Indoor air concentrations (μg/m³).

Site	Households, no.	Year, season	Time	Room	*n*	Mean	SE	Geom mean	Percentile 25	50	75	90	95	Max
LA3	Unweighted data	1987, winter	Day	LR	36	9.9	1.4	7.3	4.2	7.1	13	19	32	44
			Day	Kit	38	11	2.6	6.5	3.7	7.5	12	21	30	97
			Night	Kit	36	15	2.2	9.7	5	11	18	40	46	53
LA4	Unweighted data	1987, summer	Day	LR	40	6.5	0.9	4.9	2.8	4.6	9.4	15	20	24
			Day	Kit	38	5.5	0.8	4.4	2.5	4.7	6.7	9.9	14	28
			Night	Kit	37	6.5	1.2	4.4	2.3	4.5	7.4	13	23	44
VAL	Unweighted data	1990, summer	Day	LR	30	13	4	8.1	4	8	14	19	41	96
			Night	LR	30	18	5.2	7.6	4	8	22	29	120	120
VAL	Unweighted data	1991, winter	Day	LR	29	26	4.4	17	7	16	34	62	81	86
			Night	LR	27	24	4.6	14	7	16	28	74	79	83
WDL	10,000	1990, spring	24 hr	LR	104	4.7	1.1	2.5	1.3	2.2	5.1	8.3	–	130

Abbreviations: *n*, number; SE, standard error; geom mean, geometric mean; LR, living room; kit, kitchen. LA, Los Angeles, CA; VAL, Valdez, AK; WDL, Woodland, CA.

28-day samples in the living room and main bedroom of the home for 1 year. Thirteen sets of 12-month outdoor samples were also collected over the course of the study (November 1990–February 1993). The mean indoor concentration was 8 μg/m³ ($n = 3000$ samples) compared to an outdoor mean of 5 μg/m³ ($n = 125$).

Ambient Concentrations

Benzene concentrations were reported for 586 ambient air samples collected from 10 Canadian cities (T Dann, unpublished data). The overall mean was 4.4 μg/m³, with Ottawa and Montreal ranging between 5.1 and 7.6 μg/m³. A more recent survey (T Dann and D Wang, unpublished data) found similar levels, with three rural sites ranging from 0.6 to 1.2 μg/m³.

Twenty-four-hour average benzene levels have been measured every 12th day at about 20 sites throughout California since 1986 (*31*). Statewide average annual values fluctuated between 5 and 7 μg/m³ until 1993 and 1994, when they dropped to about 4 μg/m³ (Figure 3). This decline appears to be real and may be due to one or more of several factors: *a*) the 50% reduction in hydrocarbon emissions mandated for new cars; *b*) the Stage II vapor recovery controls recently in effect; *c*) a reduction in benzene content in gasoline down to the 1% mandated in the 1990 Clean Air Act Amendments.

The California database also allows analysis of seasonal variation. A clear sinusoidal curve is apparent, with winter values about twice summer values (Figure 4). This may be due to changes in the blend of the gasoline toward greater volatility in the winter or to increased likelihood of inversions during the winter.

The mean personal, indoor, and outdoor values of benzene measured in these more recent studies are compared in Table 4.

In-Vehicle Studies

The largest study of in-vehicle benzene exposure continues to be the 200-trip study (*32*) of Los Angeles commuters carried out in the summer and winter seasons. This study found an average benzene exposure of 13 ppb (40 μg/m³) for commuters during rush hour, on the order of 5 times the concentration measured at a fixed outdoor site.

A small study in North Carolina (*33*) also showed in-vehicle concentrations 3 to 8 times background ambient levels. A second small study in Boston (*34*) resulted in passenger levels 1.5 times roadway levels on an interstate highway.

More recently, a study (*35*) of benzene levels in two-passenger vehicles during

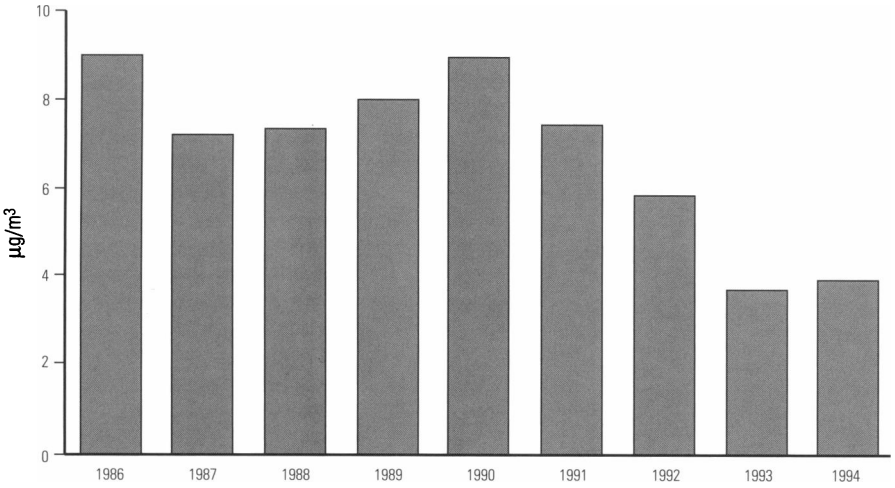

Figure 3. Ambient benzene in California, annual averages across all sites 1986–1994. Annual average outdoor benzene concentrations at about 20 sites in California. At each site, a 24-hr average is taken every 12 days. The decline in 1993 to 1994 may be due to reduced emissions from automobiles. Source: California Air Resources Board, data from 20+ cities.

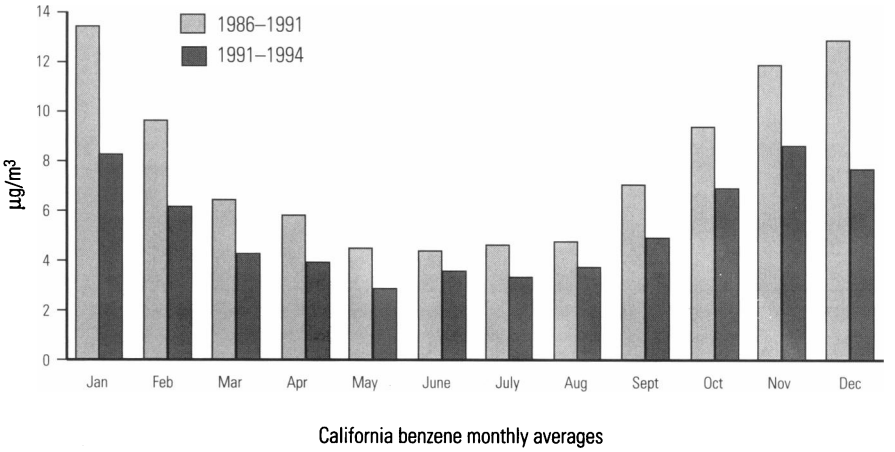

Figure 4. California benzene monthly averages. Seasonal variation in outdoor benzene at about 20 sites in California. Higher values in winter may be due to seasonally varying gasoline formulations, and perhaps to increased frequency of atmospheric inversions. The decline in benzene concentrations over the past few years is consistent over all seasons. Source: California Air Resources Board, data from all (about 20) sites.

Table 4. Mean benzene concentrations (µg/m^3) reported in recent studies.

Reference	Location	*n*	Personal	Indoor	Outdoor
Goldstein et al., 1992 (*25*)	Alaska	112	24	20	8
Sheldon et al., 1991 (*24*)	California	120	5.0	4.0	1
Heavner et al., 1994 (*26*)	Ohio	49	3.2		
Brown and Crump 1996 (*30*)	England	173		8	5
Wilson et al., 1993 (*28*)	California	161		8.3	6
Fellin and Otson, 1993 (*27*)	Canada	754		5.4	
Dann (unpublished data)	Canada	586			4
CARB 1989–1992 (*31*)	California	3000			7
CARB 1993–1994 (*31*)	California	1000			4

typical commutes in the New Jersey–New York area resulted in measured exposures of 9 to 12 µg/m^3 in suburban and turnpike conditions, and 26 µg/m^3 in the Lincoln Tunnel. The author stated that the concentrations during the commutes to New York City were about 10 times the ambient background concentration measured the same day in suburban New Jersey.

Unfortunately, none of the studies measured the benzene concentration in the gasoline used, so it is not possible to determine whether the lower concentrations in the later studies might be due to lower amounts of benzene in gasoline.

Gasoline Spill Study

A study of exposure to benzene while showering with gasoline-contaminated groundwater (*36*) was carried out in a home in North Carolina. The ground water had a measured benzene concentration of 292 µg/liter, well above the U. S. EPA's Maximum Contaminant Level of 5 µg/liter. Three 20-min showers on consecutive days resulted in peak shower-stall concentrations of 800 to 1670 µg/m^3. Bathroom concentrations reached 370 to 500 µg/m^3, and concentrations in the remainder of the house peaked (0.5–1 hr later) at 40 to 140 µg/m^3. The inhalation dose during the 20-min shower ranged from 80 to 100 µg. A dermal dose of 160 µg was also calculated, using measured breath concentrations. The combined dose of about 250 µg from the 20-min shower is roughly equal to the mean total daily inhalation dose of about 200 µg for all nonsmokers in the TEAM study (assuming 15 µg/m^3 × 14 m^3/day alveolar inspiration).

Body Burden

Benzene in the blood of 883 persons was measured (*37*) as part of the National Health and Nutrition Examination Survey (NHANES III). These blood concentrations were compared with the breath concentrations measured in about 800 persons in the TEAM studies of the 1980s. Since the TEAM study measurements were made using mixed breath, the breath values were multiplied by 10/7 to account for a dead space estimated at 30% of the volume of an inhaled breath. Theoretically, one might expect that if the two populations are comparable, the ratio of blood to alveolar air concentrations for corresponding percentiles should remain constant at the magnitude of the blood/air partition coefficient for benzene, for which several estimates ranging between 7 and 10 have been

Table 5. Breath and blood concentrations and blood/breath ratios at selected percentiles from all TEAM study sites (n = 800) and from the NHANES III population (n = 883).

Percentile	Breath, ng/liter	Blood, ng/liter	Blood/breath ratio
16th	0.63	15	24
25th	1.1	38	35
75th	15.6	166	10.6
90th	33.7	324	9.6
95th	58.4	477	8.2
99th	101	807	8.0
Median	5.6	61	11
Mean	13.1	131	10
Max	330	1880	5.7

made. However, the actual observed ratio of blood to alveolar air concentrations appears to decrease with increasing concentrations, from 24 and 33 at the 16th and 25th percentiles through a range of 11 to 8 at progressively higher percentiles (Table 5). This is similar to the observation (*38*) of a blood/breath ratio of about 20/38 for an unexposed population of nonsmoking nurses, while the ratio for an occupationally exposed cohort of smokers was about 7.7. Both these findings may be explained by the possibility suggested by Travis and Bowers (*39*) that at low concentrations, a saturable blood component (e.g., proteins) binds a limited amount of benzene, making it unavailable for distribution throughout the body or elimination in breath. Travis and Bowers estimated the capacity of the blood proteins to be 90 ng/liter, based on the observations of Perbellini et al. (*38*). They also estimated the plasma partition coefficient to be 9.0. Adding the NHANES/TEAM data to those of Perbellini et al. and adjusting the Travis/Bowers model to fit all the data, one arrives at a lower estimate of blood capacity of 30 ng/liter, and a slightly lower plasma partition coefficient of 8 (Figure 5).

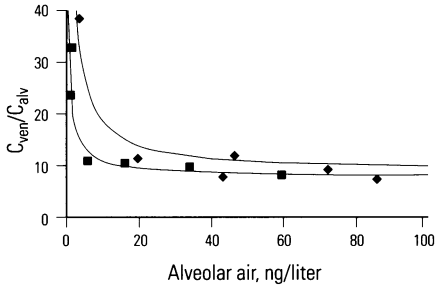

Figure 5. The upper curve is a model by Travis and Bowers (*39*) fitted to venous blood/alveolar air (C_{ven}/C_{alv}) ratios observed by Perbellini et al. (*38*). The model assumes that some benzene is bound by proteins in the blood, with a maximum capacity of 90 ng/liter. The lower curve is an adjusted model to fit both Perbellini's observations (◆) and the blood/breath ratios (■) calculated from corresponding percentiles of the NHANES blood measurements on 883 persons and the TEAM study breath measurements on about 800 persons. The adjusted model employs a somewhat smaller maximum capacity of 30 ng/liter and a slightly lower estimate of the plasma partition coefficient [8 compared to the value of 9 estimated by Travis and Bowers (*37*)].

Concentrations in Food

There were reports in the 1970s of benzene being found at ppm levels in some foods such as eggs (*40*). However, in a special study that was part of the TEAM pilot study in 1980, breath measurements before and after eating eggs showed no increase in benzene. Also, no effect on benzene levels in breath from eating eggs or any other food item could be discerned from regressions on all participants in the main TEAM study, using the participants' responses to a detailed questionnaire on food intake. It is possible that minor levels of benzene in foodstuffs could still have been present and not detected in breath due to efficient metabolization by the liver, which receives materials from the gut directly before they enter the blood stream. However, it was thought that major concentrations in food would be detectable in breath; since they were not, it was concluded that food and beverages were an unimportant pathway for benzene exposure.

Two recent studies of benzene levels in foods have confirmed that conclusion by finding negligible quantities in nearly all foods measured. In one study by the U.S. Food and Drug Administration (FDA), more than 50 foods were analyzed for benzene (*41*). Most of these were under 2 ng/g ppbw (parts per billion by weight) benzene. Exceptions included strawberry preserves (38 ng/g), taco sauce (9 and 22 ng/g), duck sauce (7 ng/g), and barbecue sauce (5 ng/g). The authors speculated that the added benzoates and ascorbates in these foods might react to form benzene; thus, if either one or the other were removed, the benzene might no longer be formed. In a second study (*42*), 57 foods were measured, with only shelled peanuts and fried eggs giving positive results, each at 30 ng/g, again far below the parts per million (ppm) levels previously reported. A recent Canadian review of benzene exposures (*43*) concluded that food and drinking water each contributed only about 0.02 µg/kg benzene per day compared to a total intake of 2.4 µg/kg per day from airborne exposures (3.3 µg/kg/day if exposed to cigarette smoke). Thus, airborne exposure accounts for 98 to 99% of total benzene intake for Canadian nonsmokers.

Discussion

The general finding from previous studies that personal exposures to benzene exceed indoor air concentrations, which in turn exceed outdoor air concentrations, has been confirmed by the more recent studies.

Two of the three personal monitoring studies mentioned above had somewhat lower mean personal exposures to benzene than had previously been reported. One such study (*24*) was in a small rural community in California, which also had a lower mean outdoor benzene value (1.2 µg/m^3) than has been previously reported. The second study (*26*) included only 3-hr exposures in the evening at home; to the extent that all other personal exposure studies included time spent in vehicles, where benzene exposures have been shown to range up to 40 µg/m^3, such a study limited to the home microenvironment might be expected to produce smaller personal exposures. Therefore, both of these studies would be expected to be at the low end of benzene exposures.

On the other hand, the outdoor concentrations of about 5 µg/m^3 in Valdez were similar to outdoor concentrations in the various TEAM study sites, but the indoor and personal concentrations (20 and 24 µg/m^3) were considerably greater than in all TEAM study sites except for Los Angeles in the winter. It may be speculated that persons in frontier-type situations make more use of gasoline-powered instruments such as chain saws, snow blowers, and snowmobiles than persons in urban communities. It may also be that the requirements for warming up automobiles for extended periods, and the larger amounts of benzene that are found in Alaskan and Canadian gasoline blends, led to higher exposures from attached garages and driving.

Considering that the TEAM Studies showed a range of benzene exposures from 7 to 29 µg/m^3 (*16*), the range observed since 1990 of 3.2 to 24 µg/m^3 provides no firm evidence as yet for a downward trend in benzene exposures.

A second finding from previous studies, that benzene levels were increased in homes with smokers, was also replicated. The new study (*26*) found a significant increase of 1.6 µg/m^3, which is less than

the increases of 3.5 and 4.5 μg/m^3 found in the TEAM and West German studies (*15,23*) but represents about the same percentage increase of 50 to 67% compared to nonsmoking homes. The Windsor study (*29*) that found increased benzene concentrations in bingo halls and taverns, where smoking is prevalent, might also be viewed as confirming the effect of smoking on indoor benzene concentrations.

Several small studies replicated the findings of an earlier major study in Los Angeles that showed increased benzene exposures while driving. The later studies appeared to involve much smaller exposures but also had much smaller outdoor concentrations, so the ratio of personal exposure to outdoor concentration continued to be in the neighborhood of 5 to 10. The smaller concentrations could be due to differences in location (Los Angeles vs North Carolina and New Jersey–New York) but could also reflect reductions in the amount of benzene in the gasoline. The results of the national fuel survey carried out by the American Automobile Manufacturers Association (*44*) indicate that the goal of 1% benzene in gasoline set by the 1990 Clean Air Act Amendments has been very nearly met, with the average for premium, intermediate, and regular gasoline for the winter of 1994 to 1995 being 0.9, 0.9, and 1.1% by volume, respectively. This is a considerable reduction compared to the 2 to 3% levels that were probably common during the large California in-vehicle study. However, it should also be noted that the amount of benzene in the exhaust may be related only weakly to the amount of benzene in the gasoline. One study (*45*) indicated that exhaust benzene remained unchanged at about 5% of total hydrocarbon emissions whether the gasoline burned contained 1 or 3% benzene by volume.

A large number of food groups were tested but found to contain negligible amounts of benzene. This corroborated the conclusions of the TEAM studies, which found no evidence of food contributions to body burden of participants.

Although nearly all the studies reviewed here have been more in the nature of confirmatory studies rather than breaking new ground, the study of benzene exposures while showering in gasoline-contaminated water presented new data of considerable value. The 20-min exposure from this source was the same order of magnitude as a full day's exposure to benzene for a typical nonsmoker. However, a smoker (of more than five cigarettes a day) using the same gasoline-contaminated water would still get most of his or her exposure through smoking—an indication of the extensive exposure encountered by some 43 million U.S. citizens. Since the number of persons affected by such spills is very small, the effect on the national exposure budget for benzene is also very small.

Apart from these presumably very rare gasoline spill situations, there may be a larger number of cases where well water is contaminated by benzene at low concentrations. A number of studies have reported finding benzene at levels on the order of 5 ng/liter (ppb) in surface and well waters. However, these levels correspond to a daily intake of < 10 ng benzene, assuming 2 liters of water drunk daily. This amount is only 0.5% of the average daily intake for nonsmokers of 200 ng from air. Thus, it is concluded that the effect of contaminated water on total benzene intake is negligible.

It may fairly be asked whether any of the differences observed in various studies at different locations and times are dependent on the different methods employed. The TEAM studies employed Tenax-GC with active pumping, as did the later Woodland and Valdez studies; thus, all the studies using personal monitors used very similar or identical methods.

The indoor air studies in England and Canada employed passive (diffusive) samplers with extended monitoring periods. The English investigators performed a number of tests on the effect of extended sampling on the net uptake of different VOCs by the Perkin-Elmer sorbent tubes containing Tenax-TA. They found that the more volatile VOCs such as benzene and toluene had net diffusive uptakes that declined over time, probably because of back diffusion off the tubes. For sorbent tubes exposed to a concentration of 2500 μg/m^3 toluene, the diffusive uptake rate declined to 71% of the ideal after 7 days and 54% after 28 days. For benzene, the net diffusive uptake was 30% after 28 days. For the less volatile compounds such as xylenes, decane, and trimethylbenzenes, the sampling rate stayed nearly constant over the 28-day period. Therefore, given a month-long sampling period, an average uptake rate can be chosen for any given chemical; however, for chemicals more volatile than the xylenes, this rate will only be an average value from a declining curve. This means that the early part of the sampling period for these volatile compounds may be underrepresented because of back diffusion losses from the substrate. However, if the average sampling rate is correctly chosen, this would not cause a bias, only greater variability than exists in fact.

The Canadian investigators used commercial samplers with a charcoal sorbent (the 3M organic vapor badge), followed by solvent desorption using carbon disulfide (CS_2). Carbon disulfide is well known to have a contamination problem with benzene; however, the Canadian investigators developed their own methods for cleaning the CS_2, and report no serious problems with contamination. It is not clear whether they used the ideal sampling rate for benzene or determined an effective sampling rate for the 1-week sampling period.

Both Tenax and charcoal have problems with nonzero background benzene concentrations. The Tenax must be carefully cleaned to avoid such problems. The early TEAM studies had high and variable backgrounds of benzene equivalent to about 5 ± 3 μg/m^3 on the Tenax cartridges. This would lead to decreased precision, although since average backgrounds were subtracted from each raw datum, it is not clear whether any bias remained. The later TEAM studies reduced backgrounds to the equivalent of about 1 ± 0.5 μg/m^3, reducing the uncertainty in the estimated exposures considerably. The charcoal badges also have high backgrounds of benzene; however, the extended sampling period should have provided sufficient benzene to reduce the background effect. Therefore it is unlikely that a significant bias or lack of precision has affected the personal, indoor, or outdoor air concentrations of benzene.

The initial breath measurements in the TEAM New Jersey study of 1981 employed Tedlar (Nutech Corp., Durham, NC) bags stored in a van. Because of the possibility that exhaust vapors had penetrated the Tedlar bags, future studies passed pure helium over the bags at positive pressure to remove this possible source of contamination. Both higher exposures and higher breath concentrations were noted in this period, but about 45% of the New Jersey participants were smokers compared to 22% of the California participants; thus the higher breath concentrations observed in the New Jersey participants may well have been due to the higher smoking rate.

REFERENCES

1. Wallace LA. The TEAM Study: Summary and Analysis. Vol 1. EPA 600/6-87/002a. NTIS PB 88-100060. Washington:U.S. Environmental Protection Agency, 1987.
2. Zweidinger R, Erickson M, Cooper S, Whitaker D, Pellizzari ED, Wallace LA. Direct Measurement of Volatile Organic Compounds in Breathing-zone Air, Drinking Water, Breath, Blood, and Urine. NTIS PB-82-186-545. Washington:U.S. Environmental Protection Agency, 1982.
3. Wallace LA, Zweidinger R, Erickson M, Cooper S, Whitaker D, Pellizzari ED. Monitoring individual exposure: measurement of volatile organic compounds in breathing-zone air, drinking water, and exhaled breath. Environ Int 8:269–282 (1982).
4. Wallace LA, Pellizzari ED, Hartwell T, Rosenzweig R, Erickson M, Sparacino C, Zelon H. Personal exposure to volatile organic compounds. I: Direct measurement in breathing-zone air drinking water, food, and exhaled breath. Environ Res 35:293–319 (1984).
5. Wallace LA, Pellizzari ED, Hartwell T, Sparacino C, Sheldon L, Zelon H. Personal exposures, indoor-outdoor relationships and breath levels of toxic air pollutants measured for 355 persons in New Jersey. Atmos Environ 19:1651–1661 (1985).
6. Wallace LA, Pellizzari E, Hartwell T, Whitmore R, Sparacino C, Zelon H. Total Exposure Assessment Methodology (TEAM) Study: personal exposures, indoor-outdoor relationships, and breath levels of volatile organic compounds in New Jersey. Environ Int 12:369–387 (1986).
7. Wallace LA, Pellizzari ED, Hartwell TD, Sparacino C, Whitmore R, Sheldon L, Zelon H, Perritt R. The TEAM study: personal exposures to toxic substances in air, drinking water, and breath of 400 residents of New Jersey, North Carolina, and North Dakota. Environ Res 43:290–307 (1987).
8. Pellizzari ED, Perritt K, Hartwell TD, Michael LC, Whitmore R, Handy RW, Smith D, Zelon H. Total Exposure Assessment Methodology (TEAM) Study: Elizabeth and Bayonne, New Jersey; Devils Lake, North Dakota; and Greensboro, North Carolina. Vol II. Washington:U.S. Environmental Protection Agency, 1987.
9. Pellizzari ED, Perritt K, Hartwell TD, Michael LC, Whitmore R, Handy RW, Smith D, Zelon H. Total Exposure Assessment Methodology (TEAM) Study: selected communities in northern and southern California. Vol III. Washington:U.S. Environmental Protection Agency, 1987.
10. Wallace LA, Pellizzari ED, Hartwell TD, Whitmore R, Perritt R, Sheldon LS. The California TEAM study: breath concentrations and personal exposures to 26 volatile compounds in air and drinking water of 188 residents of Los Angeles, Antioch, and Pittsburg, CA. Atmos Environ 22:2141–2163 (1988).
11. Pellizzari ED, Michael LC, Perritt R, Smith DJ, Hartwell TD, Sebestik J. Comparison of Indoor and Outdoor Toxic Air Pollutant Levels in Several Southern California Communities. Final Report, Contract 68-02-4544. Research Triangle Park, NC:U.S. Environmental Protection Agency, 1988.
12. Wallace LA, Nelson WC, Ziegenfus R, Pellizzari E. The Los Angeles TEAM study: personal exposures indoor–outdoor air concentrations, and breath concentrations of 25 volatile organic compounds. J Expos Anal Environ Epidemiol 1(2):37–72 (1991).
13. Lioy PJ, Wallace LA, Pellizzari ED. Indoor/outdoor and personal monitor and breath analysis relationships for selected volatile organic compounds measured at three homes during New Jersey TEAM—1987. J Expos Anal Environ Epidemiol 1(1):45–61 (1991).
14. Entz R, Thomas K, Diachenko G. Residues of volatile halocarbons in food using headspace gas chromatography. J Agric Food Chem 30:846–849 (1982).
15. Wallace LA, Pellizzari ED, Hartwell T, Perritt K, Ziegenfus R. Exposures to benzene and other volatile organic compounds from active and passive smoking. Arch Environ Health 42:272–279 (1987).
16. Wallace LA. The exposure of the general population to benzene. Cell Biol Toxicol 5:297–314 (1989).
17. Wallace LA. The Total Exposure Assessment Methodology (TEAM) Study: an analysis of exposures, sources, and risks associated with four chemicals. J Am Coll Toxicol 8:883–895 (1989).
18. Wallace LA, Pellizzari ED, Hartwell TD, Davis V, Michael LC, Whitmore RW. The influence of personal activities on exposure to volatile organic compounds. Environ Res 50:37–55 (1989).
19. Wallace LA. Major sources of benzene exposure. Environ Health Perspect 82:165–169 (1989).
20. Wallace LA. Major sources of exposure to benzene and other volatile organic compounds. Risk Anal 10:59–64 (1990).
21. Thomas KW, Pellizzari ED, Clayton CA, Perritt RL, Dietz RN, Goodrich RW, Nelson WC, Wallace LA. Temporal variability of benzene exposure for residents in several New Jersey homes with attached garages or tobacco smoke. J Expos Anal Environ Epidemiol 3:49–73 (1993).
22. Lebret E, Van de Weil HJ, Noij D, Boleij JSM. Volatile hydrocarbons in Dutch homes. Environ Int 12(1–4):323–332 (1986).
23. Krause C, Mailahn W, Nagel R, Schulz C, Seifert B, Ullrich D. Occurrence of volatile organic compounds in the air of 500 homes in the Federal Republic of Germany. In: Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Vol 1 (Seifert B, Esdorn H, Fischer M, Ruden H, Wegner J, eds). Berlin:Institute for Soil, Water, and Air Hygiene, 1987; 102–106.
24. Sheldon LS, Clayton A, Jones B, Keever J, Perritt R, Smith D, Whitaker D, Whitmore R. Indoor Pollutant Concentrations and Exposures. Final Report. Sacramento:California Air Resources Board, 1991.
25. Goldstein BD, Tardiff RG, Baker SR, Hoffnagle GF, Murray DR, Catizone PA, Kester RA, Caniparoli DG. Valdez Air Health Study. Anchorage, AL:Alyeska Pipeline Service Co., 1992.
26. Heavner DL, Morgan WT, Ogden MW. Determination of volatile organic compounds and ETS apportionment in 49 homes. Environ Int 21:3–21 (1996).
27. Fellin P, Otson R. Seasonal trends of volatile organic compounds (VOCs) in Canadian homes. In: Indoor Air '93: Proceedings of the 6th International Conference on Indoor Air Quality and Climate, Vol 1 (Jaakola JJK, Ilmarinen R, Seppänen O, eds). Espoo, Finland:Helsinki University of Technology, 1993;339–343.
28. Wilson AL, Colome SD, Tian Y. California Residential Indoor Air Quality Study. Vol 1: Methodology and Descriptive Statistics. Irvine, CA:Integrated Environmental Services, 1993.
29. Bell RW, Chapman RE, Kruschel BD, Spencer MJ. Windsor Air Quality Study: Personal Exposure Survey Results ISBN 0-7778-3492-8. Toronto:Ministry of Environment and Energy, 1994.
30. Brown VM, Crump DR. Volatile organic compounds. In: Indoor Air Quality in Homes: Part I. The Building Research Establishment Indoor Environment Study (Berry RW, Brown VM, Coward SKD, Crump DR, Gavin M, Grimes CP, Higham DF, Hull AV, Hunter CA, Jeffery IG, Lea RG, Llewellyn JW, Raw GJ, eds). London:Construction Research Communications, 1996.
31. California Air Resources Board. Ambient Air Quality Data Base. Technical Support Division. Sacramento, CA:California Air Resources Board.
32. SCAQMD. In-vehicle Characterization Study in the South Coast Air Basin. Los Angeles:South Coast Air Quality Management District, 1989.
33. Chan C-C. Commuter exposure to volatile organic compounds. PhD dissertation. Boston:Harvard University, 1990.

34. Chan C-C, Özkaynak H, Spengler JD, Sheldon L. Driver exposure to volatile organic compounds, CO, ozone, NO_2 under different driving conditions. Environ Sci Technol 25:964–972 (1991).
35. Weisel C, Lawryk NJ, Lioy PJ. Exposure to emissions from gasoline within automobile cabins. J Expo Anal Environ Epidemiol 2(1):79–96 (1992).
36. Lindstrom AB, Highsmith VR, Buckley TJ, Pate WJ, Michael L. Gasoline-contaminated ground water as a source of residential benzene exposure: a case study. J Expos Anal Environ Epidemiol 4:(2)183–196 (1994).
37. Ashley DL, Bonin MA, Carkinali L, McCraw JM, Wooten JV. Blood concentrations of volatile organic compounds in a nonoccupationally exposed US population and in groups with suspected exposure. Clin Chem 40:1401–1404 (1994).
38. Perbellini I, Faccini GB, Pasini F Cazzoli F, Pistoia S, Rosellini R, Valsecchi M, Brugnone F. Environmental and occupational exposure to benzene by analysis of breath and blood. Br J Ind Med 45:345–352 (1988).
39. Travis CC, Bowers JC. Protein binding of benzene under ambient exposure conditions. Toxicol Ind Health 5(6):1017–1024 (1989).
40. MacLeod AJ, Cave SJ. Volatile flavor compounds of eggs. J Sci Food Agric 26:351–360 (1975).
41. McNeal TP, Nyman PJ, Diachenko GW, Hollifield HC. Survey of benzene in foods by using headspace concentration techniques and capillary gas chromatography. J Assoc Organ Analyt Chem Int 76:(6)1213–1219 (1993).
42. American Petroleum Institute. Analysis of Foods for Benzene. Washington:API, 1992.
43. Government of Canada. Priority Substances List Supporting Document: Benzene. Ottawa:Health Canada, 1993.
44. American Automobile Manufacturers Association. National Fuel Surveys: Gasoline and Diesel Fuel—Winter 1995. Detroit:AAMA, 1995.
45. Sawyer, RF. Trends in auto emissions and gasoline composition. Environ Health Perspect 101(Suppl 6):5–12 (1993).